

Higher cognitive ability buffers stress-related depressive symptoms in adolescent girls

Article (Accepted Version)

Riglin, Lucy, Collishaw, Stephan, Shelton, Katherine H, McManus, I C, Ng-Knight, Terry, Sellers, Ruth, Thapar, Ajay K, Frederickson, Norah and Rice, Frances (2015) Higher cognitive ability buffers stress-related depressive symptoms in adolescent girls. *Development and Psychopathology*, 28 (1). pp. 97-109. ISSN 0954-5794

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Full article title: Higher cognitive ability buffers stress-related depressive symptoms in adolescent girls

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Acknowledgements: We thank the pupils, parent and schools who have participated in the studies and thank the field team and clinical team involved in data collection and supervision. This research has been supported by the Nuffield Foundation, UK (EDU/40065), the Jules Thorn Medical Trust and the MRC (G0802200). SC is supported by the Waterloo Foundation. AKT is funded by the National Institute for Social Care and Health Research Academic Health Science Collaboration (NISCHR AHSC).

Short title: Cognitive ability, stress and depression

Full article title: Higher cognitive ability buffers stress-related depressive symptoms in adolescent girls

Abstract: Cognitive ability and depressive symptoms tend to show an inverse association, but it is unclear whether this reflects an inherent vulnerability to depression in those of lower ability, or whether higher cognitive ability promotes resilience in the face of environmental adversity. We investigated the role of cognitive ability as a moderator of the effect of stressful life events on depressive symptoms and whether this varied by gender. Data were analysed in two adolescent datasets: one representative community sample aged 11-12 year (n=460) and one at increased familial risk of depression aged 9-17 years (n=335). In both datasets, a three-way interaction was found whereby in girls, but not boys, higher cognitive ability buffered the association between stress and greater depressive symptoms. The interaction also replicated when the outcome was a diagnosis of major depressive disorder. This buffering effect in girls was not attributable to coping efficacy. However, a small proportion of the variance was accounted for by sensitivity to environmental stressors. Results suggest that this moderating effect of cognitive ability in girls is largely attributable to greater available resources for cognitive operations which offer protection against stress-induced diminutions in cognitive processing.

Key words: depression; cognitive ability; stress; resilience; cognitive reserve

Introduction

Evidence suggests an inverse association between cognitive ability and depression both in childhood and adulthood (e.g. Collishaw, Maughan, & Pickles, 2004; Franz et al., 2011; Glaser et al., 2011; Hartlage, Alloy, Vazquez, & Dykman, 1993). Several studies show that this association persists when making statistical controls for social disadvantage (Fergusson, Horwood, & Ridder, 2005; Franz et al., 2011; Glaser et al., 2011; Hartlage et al., 1993; although see Collishaw et al., 2004 for an exception). Some research suggests this association differs by gender (Glaser et al., 2011). One proposed explanation for a direct association is that lower cognitive ability and depression share a common aetiology and have overlapping genetic and environmental risk factors (Koenen et al., 2009). An alternative possibility, which we investigate here, is that individuals with higher cognitive ability have greater cognitive reserve which serves to protect them against some of the adverse effects of stress whilst individuals of lower cognitive ability are more vulnerable to depression following stressful life events because they are less equipped to cope with them (Barnett, Salmond, Jones, & Sahakian, 2006; Koenen et al., 2009). Potential explanations of such a possibility include that there is greater capacity and efficiency of the cognitive system (i.e. greater cognitive reserve) in more able individuals which serves to protect them from some of the adverse effects of stress (see below). Another is that more cognitively able individuals may have greater capacity to profit from information acquired as a result of stressful situations, find meaning in them and positively reframing them, tendencies which have been associated with stress resilient outcomes (Southwick, Vythilingam, & Charney, 2005).

Evidence for an association between stress and depression is strong (Hammen, 2005) with stressful life events exerting a modest causal effect on the risk for major depressive disorder (Kendler & Gardner, 2010). Stress appears to have a more important role in precipitating first episodes rather than recurrent episodes of depression (Kendler, Thornton, & Gardner, 2000; Monroe & Harkness, 2005; NICE, 2005). Adolescence is a period of vulnerability for depression with first episodes often emerging at this time and the prevalence of depressive symptoms increasing markedly, particularly in girls. Adolescent depressive disorder and symptoms are associated with a range of contemporaneous and long-term difficulties including suicide attempts, hospitalisation, and impaired social, occupational and inter-personal functioning (e.g. Angold, Costello, Farmer, Burns, & Erkanli, 1999;

Weissman et al., 1999). It has also been suggested that stress experienced during adolescence can have long-lasting effects on the development of brain areas involved in the regulation of stress (Lupien, McEwen, Gunnar, & Heim, 2009). Taken together, these observations suggest that adolescence is an important period during which to assess the relationship between stress, cognitive ability and depression.

Stress impairs processing in aspects of cognition that make important contributions to cognitive ability including memory and learning (Kim & Diamond, 2002). Stressful life events are associated with poorer working memory performance, which may be due to interference from thoughts and memories of the stressful event with the on-task demands for cognitive resources (Klein & Boals, 2001). Moreover, chronic imposed stress (exam preparation) and perceived stress are associated with impairments in attentional set-shifting and functional connectivity of the dorsolateral prefrontal cortex with areas of the fronto-parietal attention network (Liston, McEwen, & Casey, 2009). Similar behavioural results (attentional set-shifting difficulties) have been reported in rodents following chronic restraint stress and this is associated with reductions in dendritic spine density and arborisation in medial prefrontal cortex (Liston et al., 2006). The implication of these findings as relates to depression is that, following a stressor, those with higher cognitive ability may have greater cognitive resources to more efficiently process the consequences of stress. These cognitive resources may include ‘hardware’ explanations such as greater functional capacity and efficiency (Gray, Chabris, & Braver, 2003; Hasher, Hasher, & Zacks, 1979; van den Heuvel, Stam, Kahn, & Hulshoff Pol, 2009) or more adaptive interpretations of stress (e.g. positive explanatory style, finding meaning in adversity) (Southwick et al., 2005). Indeed various authors suggest that higher cognitive ability is associated with more positive social, academic and mental health outcomes following a range of stressors including adverse life events (Masten et al., 1999) and recurrent depression in a parent (Pargas, Brennan, Hammen, & Le Brocque, 2010; Tiet et al., 1998).

Existing research gives some indication that the association between cognitive ability and depression may vary by gender. Some research has indicated a female-specific association between higher childhood cognitive ability and fewer depressive symptoms (Hatch et al., 2007). Others have found

higher childhood cognitive ability to predict fewer depressive symptoms for girls but more depressive symptoms for boys at 17 years with no indication of gender differences earlier in adolescence (Glaser et al., 2011). Gender differences in associations between cognitive ability and depressive symptoms may become clearer when stress is included in the investigation of this association.

Higher prevalence rates of depression in girls compared to boys are well documented from mid-puberty onwards (Angold, Costello, & Worthman, 1998; Costello, Erkanli, & Angold, 2006; Green, 2005). Two explanations for the development of these gender differences are, firstly, that girls experience more negative life events than boys (Thapar, Collishaw, Pine, & Thapar, 2012), and secondly, that girls are more likely than boys to experience depression following these stressors, because of negative coping strategies (Hankin & Abramson, 2001). Evidence from both animal and human studies suggests that females may perceive social stress as more stressful (Juster et al., 2011) and be more vulnerable to depression following social stress than males (McCormick & Mathews, 2007; Stroud, Salovey, & Epel, 2002). There is therefore evidence that girls are more susceptible to depressive symptoms than boys both following stress and if they have lower cognitive ability. In light of our suggestion that cognitive ability may buffer the effects of stress on depressive symptoms, this may suggest that such a buffering effect is greater in girls. Specifically, following negative life events, lower cognitive ability in girls may lead to an increased risk of depressive symptom via cognitive vulnerabilities such as coping efficacy and greater sensitivity to environmental stressors.

The aim of this study was to test the potential buffering effects of cognitive ability in the association between stress and depressive symptoms. Following some evidence that associations between cognitive ability and depressive symptoms may differ between high- and low-risk samples (Pargas et al., 2010), we included both types of sample in our study. Two datasets were analysed to assess this question: a representative community based sample of 11 to 12 year olds (The School Transition & Adjustment Research Study: STARS) and a sample of the adolescent offspring of depressed parents aged 9 to 17 (The Early Prediction of Adolescent Depression Study: EPAD). Our main research hypotheses were: (1) cognitive ability would not be associated with depressive symptoms when controlling for stress; (2) cognitive ability would buffer the effect of stress on depressive symptoms;

and, (3) the buffering effect of cognitive ability would be stronger in girls than boys. We also explored whether coping efficacy and sensitivity to environmental stressors were mediators of the predicted buffering effect of cognitive ability on the association between stress and depressive symptoms.

Method

Samples

The STARS sample consists of data collected from pupils in year 7 (age 11-12) attending ten mixed, non-selective secondary schools, in South-East England, UK. The schools were selected as their intake was representative of the English and Welsh population in terms of the proportion of pupils who were from economically disadvantaged households (indexed by eligibility of free school meals) and who had Special Educational Needs. The proportion of pupils from black and minority ethnic backgrounds and who did not have English as a first language was slightly higher than the general population, reflecting the inclusion of London schools. The study protocol was reviewed and approved by the university ethics committee. At each assessment, parents were given the opportunity to opt their children out of the study and informed pupil consent was obtained. Questionnaire data were collected at two stages: a postal survey during May 2012 (N=750; overall response rate=35%) and an in-school assessment during November 2012 (1712; overall response rate=87%). Cognitive ability data were collected in September of that year for six of the participating schools (N=1243). Of the 750 pupils for whom postal survey data was available, 663 (88%) completed the in-school assessments (reasons for non-completion were: pupil having left the school (N=27); parents withdrawing the pupil from the study (N=24); pupil absent from school (N=18); school withdrawing from study (N=17); pupil withdrawing themselves from the study (N=1)). Cognitive ability data were available for 460 (61% of those with baseline data), which formed our analysed sample (228 boys). The analysed sub-sample with complete data did not differ from the rest of the sample in terms of depressive symptoms ($t(1646)=1.73, p=.08$) or negative life events ($t(743)=-.36, p=.72$), but had higher cognitive ability scores ($t(1241)=-4.37, p<.001$). Mean age was 11.24 years ($SD=.43$), modal

pubertal status (Petersen, Crockett, & Richards, 1988) was late pubertal (range pre-pubertal to late pubertal) at the in-school assessment.

The EPAD data consists of 337 children aged 9-17 of parents with a history of recurrent unipolar depression. Parents were recruited from general practices in South Wales UK (78%), from a previous database of adults with recurrent unipolar depression (19%) and from advertisements in primary care (3%). Families were excluded if parents had a previous bipolar or psychotic diagnosis, were not biologically related to the child, or the adolescent had an IQ < 50. A full description of the sample is given by Mars et al. (2012). Two families were later excluded as the affected parent was re-diagnosed with bipolar disorder. Data analysed in this report came from the first stage of the study. The analysed sample consisted of 335 offspring (139 boys). Mean age was 12.39 ($SD=2.02$), modal pubertal status (Petersen et al., 1988) was late pubertal (range pre-pubertal to post-pubertal).

As expected, the two datasets differed on a number of family stressors. In the EPAD sample, 28.8% came from single parent households, 16.0% had mothers with no formal educational qualifications and 13.6% had a family income of below £10,000. In STARS, 14.7% came from single parent households, 4.7% had mothers with no formal educational qualifications and 8.5% had a family income of below £10,000.

Measures

Depression. Depressive symptoms in the STARS data were measured by self-report using the Short Mood and Feeling Questionnaire (Angold, Costello, Messer, & Pickles, 1995). The scale consists of 13 items designed to cover core symptoms of DSM-III-R depression (American Psychiatric Association, 1987) for children and adolescents. Items ask about symptoms during the past 3-months on a 3-point scale: true (2); sometimes true (1) and not true (0). Items are summed to produce a total score (possible range 0-26). A clinical cut point of 11 has been proposed for this measure (Angold et al., 1995; Angold, Erkanli, Silberg, Eaves, & Costello, 2002). Internal reliability was $\alpha=.89$, comparable to that reported by Angold et al. (1995) of $\alpha=.85$.

Depressive symptoms in the EPAD data were measured by parent and child ratings using the Child and Adolescent Psychiatric Assessment (CAPA; Angold & Costello, 2000), a semi-structured

diagnostic interview used to assess depressive disorders, as well as other psychiatric disorders. The total number of DSM-IV major depressive symptoms (possible range 0-9; American Psychiatric Association, 1994) was based on combined scores, whereby a symptom was coded as present if endorsed by either child or parent. For analyses focusing on Major Depressive Disorder (MDD), adolescents were classified as affected if this diagnosis was present at any of the 3 assessment stages (follow-up period 29 months, $SD=5.39$) of the EPAD study (7 boys; 30 girls met these criteria).

Cognitive ability. Cognitive ability in the STARS data was measured by the Cognitive Abilities Test (CAT3; Lohman et al., 2001), a standardised assessment which measures verbal reasoning, quantitative reasoning and non-verbal reasoning. The three scores were averaged to produce a cognitive ability score with higher scores indicating higher ability. CAT scores have been found to be highly reliable in terms of test-retest correlations and internal consistency estimates, and have strong predictive validity with later school performance (Strand, 2006). Cognitive ability in the EPAD data was analysed using the 10 subscales of the Wechsler Intelligence Scale for Children, which has also shown high reliability and validity (WISC-IV; Wechsler, 2003).

Stress. Stress was measured by negative life events for both the datasets using a version of the Life Events Checklist (Johnson & McCutcheon, 1980), listed in Appendix 1. The checklist for each consisted of 19 binary items (e.g. parents nagging/picking on you more, losing a close friend through arguments, doing badly in school work), which may have happened in the past year, summed to give a total score (possible range 0-19, higher scores indicating more negative life events). Life events were measured using child report for STARS and parent and child reports (coded as present if endorsed by either rater) for EPAD. Life events which did not result from characteristics of the participant (e.g. death of a grandparent) were coded as behaviour-independent (Appendix 1).

Additional measures for mediated moderation analysis.

Coping efficacy. Coping efficacy was measured in STARS using the Pearlin Mastery Scale (Pearlin & Schooler, 1978). Participants were asked to what extent seven statements describe themselves (e.g. There is really no way I can solve some of the problems I have; I have little control over the things that happen to me; I often feel helpless in dealing with the problems of life) on a 5-

point scale ranging from strongly disagree (1) to strongly agree (5). Items reverse scored and summed to produce a total score (possible range 7-35, higher scores indicating greater coping efficacy, Chronbach's $\alpha=.60$).

Coping efficacy was measured in EPAD using The General Self-Efficacy Scale (Schwarzer & Jerusalem, 1995). Participants were asked to what extent ten statements describe themselves (e.g. I can always manage to solve difficult problems if I try hard enough; I am confident that I could deal efficiently with unexpected events; I can remain calm when facing difficulties because I can rely on my coping abilities) on a 4-point scale ranging from not at all true (1) to exactly true (4). Items are summed to produce a total score (possible range 10-40, higher scores indicating greater coping efficacy, Cronbach's $\alpha=.85$).

Sensitivity to environmental stressors. Sensitivity to environmental stressors was measured by sensitivity to the environment in STARS and perceived stress in EPAD. Sensitivity was measured in STARS using a self-report version of the Highly Sensitive Child Questionnaire, Short Form (Aron & Aron, 1997). The scale contained eight items (e.g. I am annoyed when people try to get me to do too many things at once; I don't like it when things change in my life; I don't like loud noises) on a 7-point scale: not at all (1); moderately (4); extremely (7). Items are summed to produce a total score (possible range 8-56, higher scores indicating greater sensitivity to the environment, Cronbach's $\alpha=.63$).

Perceived stress was measured in EPAD using a version of the Life Events Checklist (see above, Johnson & McCutcheon, 1980). Participants were asked to rate the severity of each event which they stated has having occurred on a 5-point scale from very unpleasant (1) to very pleasant (5). Scores were reversed so that a higher score indicates greater perceived severity and a score of 0 allocated if the event was not experienced. Items were summed to produce a total score (possible range 0-95, higher scores indicating greater perceived stress).

Statistical analysis

The association between cognitive ability and depressive symptoms was analysed using a series of multiple regression analyses with centred independent variables (Cohen, Cohen, Stephen, & Leona,

2003). The dependent variable was depressive symptoms. The first model tested for a direct association, with cognitive ability as a single predictor variable. Stress was controlled for in the second model and the interaction of cognitive ability x stress was added to the third model. Finally, the fourth model tested for moderation by stress and gender, which included gender, all possible additional two-way interaction terms and the interaction of cognitive ability x stress x gender. Gender was coded girls = 0, boys = 1. Significant interactions were followed-up with simple slopes analyses (Dawson & Richter, 2006) where low and high cognitive ability and stress were plotted as the sample mean \pm one standard deviation. Results are presented for the STARS data followed by the EPAD data unless otherwise stated. A final set of analyses exploring potential mediators of the hypothesised interaction between cognitive ability, stress and depression were carried out with mediated moderation using Process, model 8, in SPSS (Hayes, 2013). This tested whether the hypothesised moderation of the association between stress and depressive symptoms by cognitive ability, was attributable to coping efficacy or sensitivity to environmental stressors.

Results

Table 1 presents descriptive data and correlations for the whole sample and separately for each gender. Mean levels of negative life events were higher in EPAD than STARS (1.21 and 3.40 respectively). The proportion of participants meeting either the clinical cut-point for depression (STARS) or diagnostic criteria for depressive disorder (EPAD) was also higher in EPAD reflecting the fact that EPAD was selected on the basis of elevated familial risk for depression (13.6% in EPAD; 7.6% in STARS based on the suggested clinical cutpoint, Angold et al., 1995; Angold et al., 2002). Mean cognitive ability scores were 103.51 in STARS and 94.92 in EPAD. Independent-sample *t*-tests showed girls had higher levels of depressive symptoms than boys in both datasets, higher cognitive ability than boys in the EPAD data and more negative life events and sensitivity than boys in the STARS data. There were no gender differences for coping efficacy or independent life events. Bivariate correlations for each sample showed that depressive symptoms were associated with negative life events in both datasets ($r=.34, p<.0001$; $r=.32, p<.0001$) and with cognitive ability in the EPAD data ($r=-.13, p=.02$) but not the STARS data ($r=-.01, p=.88$). Negative life events were also

associated with cognitive ability in the EPAD data ($r=-.16, p=.004$) but not the STARS data ($r=.04, p=.37$).

Direct associations between cognitive ability and depression

Before presenting results relating to the hypothesised interaction between cognitive ability, stress and depressive symptoms, we present findings of 1) the direct association between cognitive ability and depression and 2) this association when controlling for stressful life events. Findings testing the main effect of cognitive ability on depressive symptoms are presented in Table 2 (Models 1 and 2). There was an initial association between higher cognitive ability and fewer depressive symptoms for the EPAD data ($\beta=-.12, p=.04$) but not the STARS data ($\beta=-.01, p=.80$). When controlling for stress, there was no association in either dataset ($\beta=.0004, p=.92; \beta=-.07, p=.21$). In addition to the results presented in Table 2, there was no moderation by gender for either the direct association (gender-by-cognitive ability interaction terms: $\beta=.06, p=.40; \beta=.06, p=.41$) or when controlling for stress (gender-by-cognitive ability interaction terms: $\beta=.06, p=.37; \beta=.07, p=.28$).

Cognitive ability buffering the effect of stress on depressive symptoms and disorder

Findings testing the hypothesised buffering effects of cognitive ability on the association between stress and depressive symptoms are also presented in Table 2. Initial analyses (Model 3) showed no evidence of cognitive ability moderating the effects of stress on depressive symptoms, (interaction terms: $\beta=-.04, p=.34; \beta=-.07, p=.18$) with a main effect of stress ($\beta=.33, p<.0001; \beta=.29, p<.0001$) but not cognitive ability ($\beta=.004, p=.94; \beta=-.07, p=.21$) on depressive symptoms. Further analyses revealed a three-way interaction between stress, cognitive ability and gender (Model 4; three-way-interaction terms: $\beta=.13, p=.03; \beta=.17, p=.01$).

Simple slopes analyses were used to follow-up the three-way interaction and revealed a buffering effect of higher cognitive ability for girls (Figure 1, panels a and b) with a significantly stronger association between stress and depressive symptoms for girls of lower cognitive ability compared to girls of higher cognitive ability ($t=-2.18, p=.03$ STARS; $t=-2.86, p=.01$ EPAD). For boys, there was no significant difference between lower and higher cognitive ability in the association

between stress and depressive symptoms (Figure 1, panels d and e; $t=1.10$, $p=.27$ STARS; $t=1.23$, $p=.22$ EPAD). Associations were stronger for girls of lower cognitive ability compared to boys of lower cognitive ability ($t=-2.38$, $p=.02$ STARS; $t=-1.96$, $p=.05$ EPAD). In contrast, there was some indication that associations were stronger for boys of higher cognitive ability compared to girls of higher cognitive ability in the high risk sample only ($t=-.88$, $p=.38$ STARS; $t=1.88$, $p=.06$ EPAD). There was no difference between slopes comparing boys of high cognitive ability and girls of low cognitive ability ($t=-.31$, $p=.76$ STARS; $t=.58$, $p=.56$ EPAD) or comparing boys of low cognitive ability and girls of high cognitive ability $t=-1.16$, $p=.25$ STARS; $t=-.52$, $p=.61$ EPAD. In summary, higher cognitive ability was found to buffer the association between stress and depressive symptoms in girls, but not in boys.

Logistic regression analyses (EPAD only) showed that the three-way cognitive ability x stress x gender interaction replicated when a diagnosis of MDD was the outcome variable ($Exp(B)=1.04$, $p=.04$), along with main effects of stress ($Exp(B)=1.36$, $p=.002$) and gender ($Exp(B)=.058$, $p=.02$), but not cognitive ability ($Exp(B)=.97$, $p=.09$), and no significant two-way interactions (cognitive ability x stress: $Exp(B)=.99$, $p=.38$; cognitive ability x gender: $Exp(B)=.86$, $p=.05$; stress x gender: $Exp(B)=1.55$, $p=.14$); results for girls are shown in Figure 1 panel c and for boys in Figure 1 panel f.

Behaviour-independent life events

Tests of (i) the direct associations between cognitive ability (Models 1 and 2) and (ii) the buffering effect of cognitive ability on the association between stress and depressive symptoms (Models 3 and 4) both replicated when restricting the measurement of stress to behaviour-independent life events (Table 3). Restricting the analysis to independent life events outside the control of the individual (Appendix) allowed us to rule out the possibility that the interaction was due to effects of cognitive ability on stress exposure.

Mediated Moderation analyses

Follow-up mediated moderation analyses were conducted to test whether the observed moderating effect of cognitive ability on the association between stress and depressive symptoms for girls, was

accounted for by coping efficacy or sensitivity to environmental stressors. There was no evidence of mediated moderation for coping efficacy in either sample ($b = -.001$, $SE = .004$, $CI [-.01, .01]$ STARS; $b = -.001$, $SE = .001$, $CI [-.004, .0002]$ EPAD). There was some indication of a mediated moderation for sensitivity to environmental stressors in both samples ($b = -.01$, $SE = .004$, $CI [-.02, -.001]$ STARS; $b = -.002$, $SE = .001$, $CI [-.01, -.0001]$ EPAD). That is, there was a small but significant indirect effect of the interaction between cognitive ability and stress on depressive symptoms through environmental sensitivity. This suggests that higher cognitive ability in girls may be associated with fewer depressive symptoms partly via decreased sensitivity to environmental stressors when exposed to stressful life events. However, this only accounted for a small proportion of the observed interaction.

Discussion

We set out to examine the relationship between cognitive ability and depressive symptoms, with a specific prediction that higher cognitive ability may promote resilience to stressful life events. We examined the role of cognitive ability as a moderator of the association between stressful life events and depressive symptoms in two adolescent samples where the degree of stress exposure was expected to differ. Based on the suggestion that individuals of lower cognitive ability may be less equipped to cope with stressful life events and thus be more vulnerable to their depressogenic effects (Barnett et al., 2006; Koenen et al., 2009), we predicted that cognitive ability would not be associated with depressive symptoms when controlling for stress, but that higher cognitive ability would buffer the effect of stress on depressive symptoms. Given the increased vulnerability to depression following stress found in adolescent girls (Hankin & Abramson, 2001), we also predicted that the buffering effect of cognitive ability would be stronger in girls than boys.

The data supported our first hypothesis that cognitive ability is not associated with depressive symptoms when controlling for exposure to stressful life events. The finding that controlling for stress attenuates associations between cognitive ability and depressive symptoms is consistent with previous work which found that social disadvantage strongly contributed to associations between mild learning disability in childhood and depressed mood in adulthood (Collishaw et al., 2004). This is important when considering the role of cognitive ability in the aetiology of depression, suggesting that

lower cognitive ability is not in itself a risk factor for depressive symptoms. Instead, reported bivariate associations between lower cognitive ability and greater depressive symptoms may in part be due to co-occurrence of low cognitive ability and greater exposure to stressors.

Results also provided support for our second hypothesis that higher cognitive ability buffers the effect of stress on depressive symptoms, although this was specific to girls. The observation that increased cognitive ability buffers against the depressogenic effects of stressful life events at least in adolescent girls is consistent with previous work which has found that those of higher cognitive ability show more positive outcomes such as academic, behavioural, social and psychiatric competence following stress (e.g. Masten et al., 1999; Pargas et al., 2010; Tiet et al., 1998). Our findings illustrated that higher cognitive ability was associated with resilience to life stress both in a sample of adolescents at increased familial risk of depression and a community sample of adolescents. Consistent with resilience work which highlights cognitive ability as a predictor of better mental health particularly in those at high familial risk (Pargas et al., 2010), we found bivariate associations between cognitive ability and depression in our sample of children of depressed parents, but not in our community sample. Nevertheless, this association did not remain once controlling for exposure to stressful life events. Thus, rather than having a direct association, higher cognitive ability appears to be a protective factor for depression in the context of stressful life events. Exposure to stressful life events can be controllable and partly dependent on behaviour (e.g. getting into a fight and being injured) or uncontrollable and independent of behaviour (e.g. death of a loved one) (Kendler & Baker, 2007). Thus, it is possible that individuals influence stress exposure through their own behaviour. We tested whether results replicated when stress exposure was restricted to independent life events and found the same pattern of results. The fact that results replicated give greater confidence in our interpretation that cognitive ability modifies the influence of stress on the individual as opposed to influencing stress exposure (Hammen, 1991; Rice, Harold, & Thapar, 2003).

Our data also support our third hypothesis that the buffering effect of higher cognitive ability would be greater for girls than boys. The finding that higher cognitive ability buffers the association between stress and depressive symptoms in girls only, is somewhat consistent with previous studies showing associations between higher cognitive ability and fewer depressive symptoms in girls but not

boys (Glaser et al., 2011; Hatch et al., 2007). It is also consistent with data showing that from adolescence onwards, girls experience more social stressors and are more likely to develop depressive symptoms following social stressors (Thapar et al., 2012). We also observed suggestive evidence of an opposite effect in boys, at least in those at high risk, and this merits further investigation.

Following reports of association between cognitive re-appraisal and resilience to stress-related depression (Southwick et al., 2005) and of suggestions that such cognitive vulnerabilities result in an increased risk of depression in girls (Hankin & Abramson, 2001), we tested coping efficacy as a possible mediator for the buffering effect found in girls; however, we found no evidence of a mediated moderation. We also explored sensitivity to environmental stressors as a possible mediator of this moderation following suggestions that girls may find social stress more stressful, and be more vulnerable to depression following social stress than males (Juster et al., 2011; McCormick & Mathews, 2007; Stroud et al., 2002). We found some evidence of a mediated moderation, with higher cognitive ability in girls leading to fewer depressive symptoms under stress at least partly via reduced environmental sensitivity. Thus, girls of higher cognitive ability may be better able to process information without impairing functioning under stress, due to greater capacity and efficiency of the cognitive system compared to girls of lower cognitive ability. Nevertheless, the mediation effect was small and we encourage future work looking at other possible mechanisms for this association. In particular, our findings are consistent with ‘hardware’ interpretations that effects may be due to greater cognitive capacity and efficiency (Brewin & Smart, 2005; Ellis, 1990).

Our study has a number of strengths; particularly the use of two, independent datasets which differed on background stress levels. One sample included the offspring of depressed parents, a group known to differ from the general population on exposure to stress (Goodman & Gotlib, 1999). Findings replicated across both samples, despite differing mean levels of stressful life events, age ranges, measures of cognitive ability and measure of depressive symptoms and disorder suggesting our findings are reliable. A limitation is that we were unable to investigate the possibility of shared genetic risk between cognitive ability, stress and depression. However, the fact that the pattern of results replicated when using behaviour-independent negative life events makes it unlikely that the interaction we observe is due to life events exposure arising from the characteristics of the individual

including genetically influenced characteristics (e.g. Hammen, 1991; Rice et al., 2003). Thus, this is inconsistent with shared genetic risk for stress and either cognitive ability and depressive symptoms. An alternative possibility is that associations are due to the confounding effects of deprivation which may be associated with cognitive ability, stress and depression (Collishaw et al., 2004). However, our results replicated when we repeated our analyses controlling for socioeconomic status (indexed by free-school meals eligibility in STARS and the 2000 Standard Occupational Classification for EPAD) suggesting that this is not the case (results available from the first author). A final consideration is the inference of causality. We make a case for girls of higher cognitive ability being of lower risk of increased depressive symptoms following stress. Our results do not exclude the possibility of reverse causation, however, our results replicated for behaviour-independent life events, and cognitive ability shows high stability from childhood to old age (Deary, Whalley, Lemmon, Crawford, & Starr, 2000) and thus lower cognitive ability is a better candidate for an antecedent of, rather than a result of, depressive symptoms.

Our findings suggest that low cognitive ability is not a vulnerability factor for depressive symptoms. Instead, cognitive ability moderates the association between stress and depressive symptoms for girls, with higher cognitive ability showing a buffering effect consistent with the view that cognitive ability is one resilience promoting factor in the context of stress both in children at low and high familial risk for depression.

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Table 1:

Descriptive statistics and correlations: depressive symptoms, cognitive ability, negative life events, coping efficacy and sensitivity to environmental stressors

	Whole sample	Boys	Girls	Gender differences	Depressive symptoms	Cognitive ability	Negative life events	Coping efficacy	Sensitivity
	Mean (SD)	Mean (SD)	Mean (SD)	<i>t</i> (<i>df</i>)					
<i>STARS</i>									
Depressive symptoms	3.70 (4.35)	3.08 (3.69)	4.30 (4.83)	3.00** (446)		.04	.26***	-.24***	.28***
Cognitive ability	103.51 (11.98)	103.71 (12.48)	103.32 (11.48)	-.35 (458)	-.04		-.08	.28***	.19*
Negative life events	1.21 (1.39)	1.08 (1.20)	1.35 (1.54)	2.12* (456)	.36***	-.01		-.25***	.20**
Coping efficacy	27.24 (4.65)	27.45 (4.70)	27.04 (4.61)	-.95 (453)	-.35***	.20**	-.45***		-.07
Sensitivity to the environment	33.08 (7.65)	31.62 (7.49)	34.43 (7.57)	3.68*** (389)	.29***	.05	.16*	-.26***	
Behaviour-independent life events	.62 (.78)	.55 (.69)	.69 (.86)	1.93 (456)					
<i>EPAD</i>									
Depressive symptoms	1.69 (1.86)	1.38 (1.54)	1.91 (2.04)	2.59* (329)		-.09	.37***	-.20	.29**
Cognitive ability	94.92 (12.86)	93.22 (11.34)	96.15 (13.75)	2.05* (328)	-.17*		-.20*	.24*	-.15
Negative life events	3.40 (2.37)	3.35 (2.44)	3.44 (2.33)	.33 (316)	.29***	-.14		-.03	.79***
Coping efficacy	27.73 (4.78)	27.82 (4.74)	27.67 (4.82)	-.23 (245)	-.25**	.21*	-.14		-.06
Sensitivity: perceived stress	8.28 (8.10)	7.37 (7.50)	9.00 (8.47)	1.70 (295)	.36***	-.21**	.69***	-.09	
Behaviour-independent life events	1.25 (1.21)	1.22 (.10)	1.28 (1.26)	.46 (316)					

NB. Correlations for boys lie above the diagonal, correlations for girls lie below the diagonal. * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 2:

Associations between cognitive ability and depressive symptoms: total negative life events

	STARS					EPAD				
	Model change		β	Coefficients		Model change		β	Coefficients	
	R^2	p		B ($S.E.$)	p	R^2	p		B ($S.E.$)	p
	change					change				
<i>Direct association (Model I)</i>										
Intercept				3.71 (.21)	<.0001				1.68 (.11)	<.0001
Cognitive ability			-.01	-.004 (.02)	.80			-.12	-.02 (.01)	.04
<i>Controlling for stress (Model II)</i>										
	.11	<.0001				.08	<.0001			
Intercept				3.70(.20)	<.0001				1.69 (.10)	<.0001
Cognitive ability			.0004	.002 (.02)	.92			-.07	-.01 (.01)	.21
Stress			.34	1.04 (.14)	<.0001			.29	.23 (.04)	<.0001
<i>Moderation by stress (Model III)</i>										
	.002	.34				.01	.18			
Intercept				3.69 (.20)	<.0001				1.67 (.10)	<.0001
Cognitive ability			.004	.001 (.02)	.94			-.07	-.01 (.01)	.21
Stress			.33	1.03 (.14)	<.0001			.29	.23 (.04)	<.0001
Cognitive ability x Stress			-.04	-.01 (.01)	.34			-.07	-.004 (.003)	.18
<i>Moderation by stress and gender (Model IV)</i>										
	.03	.01				.04	.004			
Intercept				4.12 (.27)	<.0001				1.87 (.13)	<.0001
Cognitive ability			-.03	-.01 (.02)	.62			-.13	-.02 (.01)	.05
Stress			.35	1.10 (.18)	<.0001			.32	.25 (.06)	<.0001
Gender			-.10	-.90(.39)	.02			-.12	-.44 (.21)	.03
Cognitive ability x Stress			-.13	-.04 (.02)	.03			-.17	-.01 (.004)	.01
Cognitive ability x Gender			.06	.03 (.03)	.39			.07	.02 (.02)	.28
Stress x Gender			-.06	-.30 (.29)	.30			-.002	-.002 (.09)	.98
Cognitive ability x Stress x Gender			.13	.06 (.03)	.03			.17	.02 (.01)	.01

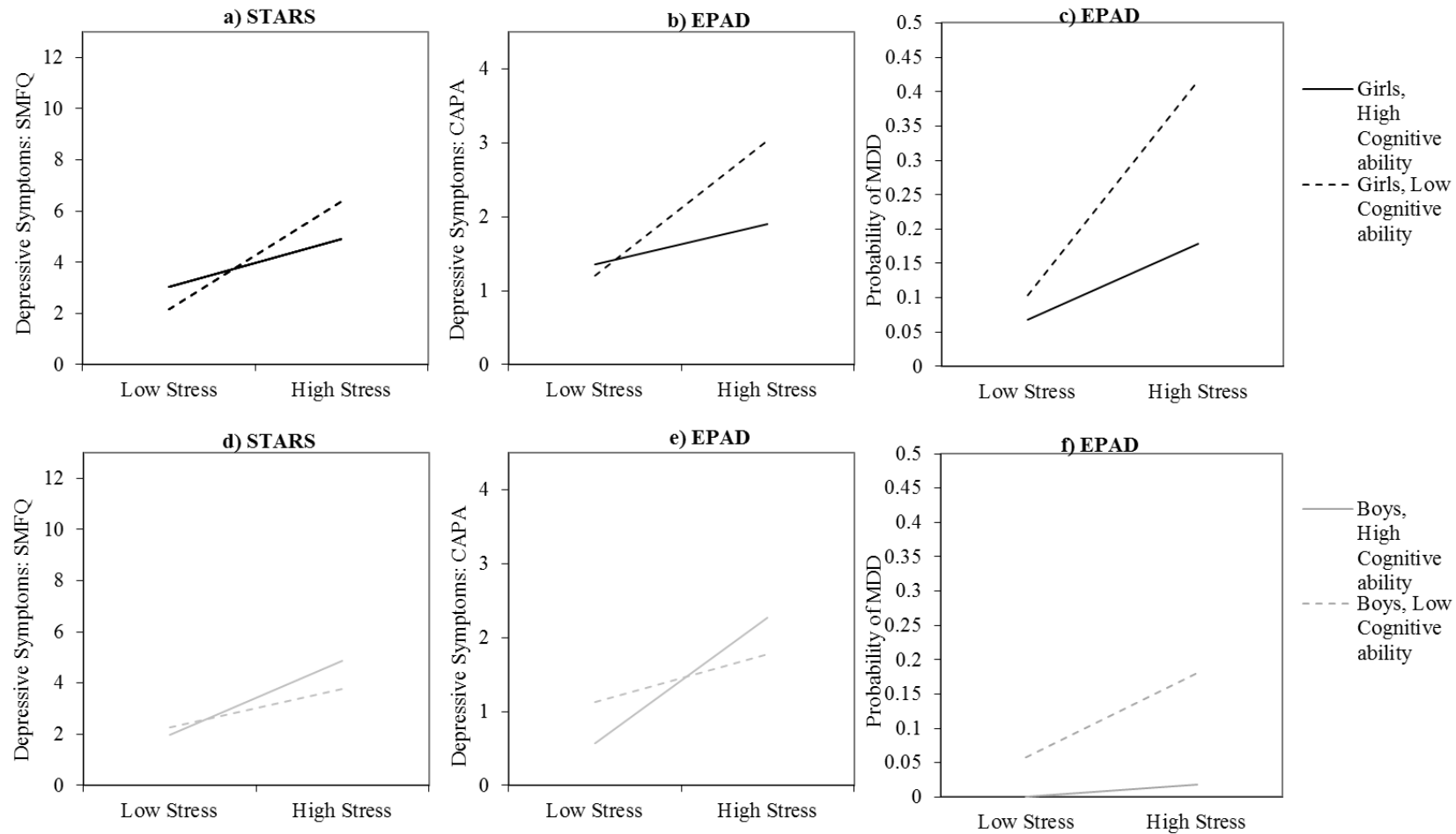
Table 3:

Associations between cognitive ability and depressive symptoms: behaviour-independent negative life events

	STARS					EPAD				
	Model change R^2 change	p	β	Coefficients B (S.E.)	p	Model change R^2 change	p	β	Coefficients B (S.E.)	p
<i>Direct association (Model I)</i>										
Intercept				3.71 (.21)	<.0001				1.68 (.11)	<.0001
Cognitive ability			-.01	-.004 (.02)	.80			-.12	-.02 (.01)	.04
<i>Controlling for stress (Model II)</i>										
	.02	.001				.01	.08			
Intercept				3.70(.20)	<.0001				1.67 (.11)	<.0001
Cognitive ability			-.02	-.01 (.02)	.67			-.11	-.02 (.01)	.06
Stress			.16	.86 (.26)	.001			.10	.16 (.09)	.08
<i>Moderation by stress (Model III)</i>										
	.01	.05				.01	.11			
Intercept				3.72 (.20)	<.0001				1.67 (.11)	<.0001
Cognitive ability			-.02	-.01 (.02)	.61			-.11	-.02 (.01)	.06
Stress			.17	.93 (.26)	.0004			.10	.16 (.09)	.08
Cognitive ability x Stress			-.09	-.04 (.02)	.05			-.09	-.01 (.01)	.11
<i>Moderation by stress and gender (Model IV)</i>										
	.03	.01				.04	.01			
Intercept				4.25 (.28)	<.0001				1.91 (.14)	<.0001
Cognitive ability			-.06	-.02 (.03)	.35			-.17	-.02 (.01)	.02
Stress			.20	1.12 (.33)	.001			.10	.16 (.11)	.17
Gender			-.13	-1.16 (.41)	.01			-.14	-.52 (.21)	.02
Cognitive ability x Stress			-.16	-.08 (.03)	.01			-.17	-.02 (.01)	.01
Cognitive ability x Gender			.06	.03 (.04)	.35			.07	.02 (.02)	.34
Stress x Gender			-.09	-.81 (.54)	.14			.04	.10 (.19)	.62
Cognitive ability x Stress x Gender			.12	.09 (.05)	.05			.14	.03 (.02)	.03

Figure 1:

Simple slopes analysis: associations between cognitive ability and depressive symptoms/MDD for girls and boys



NB. Low and high stress and cognitive ability and are plotted as the mean \pm 1SD for the two datasets. All predictor variables are centred.

Appendix 1:

Negative life events

1. Serious illness in family member*
2. Increased quarrelling between parents
3. Death of parent/brother/sister*
4. Death of grandparent*
5. Death of close friend*
6. Serious illness/injury to close friend*
7. Parent in trouble with police*
8. Parent going to prison*
9. Doing badly in an exam
10. Parents being less interested/loving
11. Parents nagging/picking on you more
12. Serious illness to you*
13. Doing badly in (school) work
14. Close friend moves away*
15. Losing a close friend through arguments
16. Death of a pet*
17. Mother losing job*
18. Father losing job*
19. Being bullied

*Coded an independent life events